The use of ultrasonography in the diagnosis and treatment of the lateral epicondylitis. Pictorial essay.

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Abstract

Lateral epicondylitis (LE) is a common orthopedic problem. It is a not life-threatening condition and does not cause severe disability, but it is a burden in everyday life. This paper focuses on this area and provides guidance on how to effectively perform US examination of the lateral part of the elbow. We will provide recent evidence on LE with particular emphasis on the role of the ultrasound in diagnosis, treatment, and disease management and we will exemplify the pathology with representative cases.

Keywords: lateral epicondylitis; ultrasound; common extensor tendon

Introduction

Lateral epicondylitis (LE) is a common orthopedic problem affecting 1-3% of adults [1]. According to the Sanders et al. population study, the incidence of LE was 3.4 per 1000 patients with a peak among individuals aged 40-49 years (7.8/1000 for male patients and 10.2/1000 per female patients) [2]. It is a not life-threatening condition and does not cause severe disability but has a burden in everyday life. Pain is often recurrent and worsens after everyday activities such as ironing, cleaning, washing windows, etc. Although the disease is called “tennis elbow”, people practicing this sport constitute a small proportion of patients presenting with this problem [2]. We aim to provide recent evidence on LE with particular emphasis on the role of the ultrasound (US) in diagnosis, treatment, and disease management. We illustrated this pictorial essay with representative US images from cases examined in our department.

Pathophysiology

LE most often affects the tendinous attachment of extensor carpi radialis brevis muscle. The extensor digitorum communis, extensor digiti minimi, and extensor carpi ulnaris also may be affected. All mentioned muscles have a common attachment called the common extensor origin (CEO) or common extensor tendon (CET) which is responsible for the extension of the wrist, fingers, and supination of the forearm [3,4]. The extensor attachments remain in close contact with the ligaments of the lateral side of the elbow. Figure 1 demonstrates the schematic anatomy of ligaments and extensor attachments.

Fig 1. Schematic anatomy of ligaments (a) and extensors attachments (b); a) yellow – lateral ulnar collateral ligament, green – lateral radial collateral ligament, blue – annular ligament; b) yellow attachment of extensor carpi radialis brevis, green – attachment of extensor carpi ulnaris, blue – enthesis of extensor digitorum communis, red – enthesis of extensor digiti minimi.
There are many theories as to what causes this enthesopathy: the inflammatory theory, the mechanical theory, the theory of the synovio-entheseal complex, as well as the autoimmune and genetic theory [5]. We, however, share the opinion that the degenerative process plays a major role in etiopathogenesis [5,7]. LE is caused by repeated microtraumas that heal ineffectively, which is the result of poor vascularization of the tendon tissue of extensor attachments [5-9]. The injuries lead to angiofibroblastic hyperplasia caused by the accumulation of fibroblasts, vascular hyperplasia, and disorganized collagen. The abnormal scar tissue leads to attachment swelling. This swollen attachment becomes more susceptible to further micro-injuries - the “vicious circle” mechanism. If the condition lasts long enough and the patient does not limit the activity of the diseased limb, the structure of the attachment may become more damaged, tears may appear, and therefore calcification may form within the tendon attachment [6,9,10].

The disease can also occur due to one-time injury - a single sudden extensor contraction at an attempt of supernatural effort, or a torsion trauma, or even after dislocation: greater damage may occur immediately (de-lamination, tearing, or even attachment breakage) [3,6,7,11-13].

**Normal US aspect**

In a quick and non-invasive way, US assessment imaging can confirm (or exclude) the diagnosis of LE. US also gives the opportunity to determine if there is a tendency to self-healing. Beyond the ability to monitor treatment, it is also used to perform treatment procedures under US guidance [4,14]. The examined elbow of a sitting patient should be flexed to 90˚ with a pronated wrist. The probe is held so that its proximally directed edge covers the surface of the lateral epicondyle of the humerus - as shown in figure 2a. By turning the probe 90˚, the cross-section can be visualized (fig 2b).

Normal extensors attachment has a typical filamentous echostructure, intermediate echogenicity, uniform thickness. Although from a practical point of view we are talking about a CEO, using high frequency US imaging the attachment fields of individual extensors - extensor carpi radialis brevis, extensor digiti minimi, extensor digitorum communis and extensor carpi ulnaris especially in transverse scans can be distinguished [15]. Figure 3 presents anatomic CEO attachment to the lateral epicondyle of the humerus.
To examine the tendon, special techniques can be used such as elastography showing differences in tissue hardness, or techniques imaging vascularization as power Doppler imaging or micro flow imaging (fig 4). Since we believe that elastography requires even more standardization, we will not focus on this issue in this paper.

US examination gives us the opportunity to assess other structures in the examined area, such as the lateral ulnar collateral ligament or the annular ligament which we demonstrate in figure 5.

**US findings in LE**

Typical findings in patients with LE are focal areas with decreased echogenicity, thickening, and alteration of the typical fibrillar echostructure. Although the typical US findings of the disease described in the literature include increased vascularity observed in the power/color Doppler study, we almost never observed increased vascularity, except for patients with spondylarthitis, which is also indicated by other authors [13,16] (fig 6). Commonly deep and anterior fibers of
the extensor carpi radialis brevis of CEO are affected [14,17].

In longstanding disease, degenerative processes such as advanced partial or even complete tears may appear (fig 7), sometimes resulting in the formation of calcification (fig 8) [17].

Enthesophytes, are small echogenic reflections (step up lesions) corresponding to calcifications, connected with the epicondyle surface and are usually located in the most proximal part of the attachment. Although they may be a consequence of a history of enthesopathy, most often the patients diagnosed with an isolated enthesophyte do not report symptoms. Sometimes we observe enthesophytes together with scar tissue resulting from minor or major lesions healing processes (fig 9).

US may help also in the differential diagnosis. Early identification of the disease may result in faster referral to a specialist or surgery to avoid permanent injury. US assessment can reveal synovial folds, radial tunnel syndrome, loose bodies, arthritis (fig 10), signs of fractures or abnormalities that may mimic arthritis, such as massive post-traumatic calcification [18] (fig 11).

**Fig 7.** a) longitudinal and b) transverse views of a partial tear within the common extensor origin (CEO); c) longitudinal and d) transverse views of a complete CEO tear. LEC – lateral epicondyle, RH – radial head.

**Fig 8.** Two cases of common extensor origin (CEO) tendinopathy with calcific deposits: a) longitudinal and b) transverse views a large calcific deposits – white open arrow - with acoustic shadow; c) longitudinal and d) transverse views of a smaller calcific deposits – white open arrow; in image “c” small enthesophyte is marked with an open arrow head.

**Fig 9.** a) and b) Two cases of enthesopathy (with symptoms) of the common extensor origin (CEO) – hypoechoic thickening altered echostructure, and enthesophytes (open arrows). The presence of enthesophytes indicates the chronic or recurrent nature of the disease: c) longitudinal view of an old CEO injury, healed with the formation of echogenic scar (section A), and calcifications (open arrow head). Open arrow - enthesophyte, d – power Doppler (same case as in c) – no increase blood flow is observed; LEC – lateral epicondyle, RH – radial head.

**Fig 10.** a) Normal fibrous plate (open arrow) in the anterior part of the humeroradial joint, HC – the humeral capitellum covered with anechoic cartilage, RH – the radial head; b) radial tunnel syndrome – white arrows – hypoechoic, swollen part of the deep branch of the radial nerve proximal to compression site, white arrowheads – compressed segment of the deep branch of the radial nerve; small black arrow on the right Frohse arcade; c) loose body – open arrow head in the humeroradial joint, HUM – posterolateral part of the distal humerus, RH – radial head, d) osteoarthritis of the elbow joint with small effusion (section A), osteophyte (asterix), and – humeral capitellum (HC) with full thickness cartilage defects. RH – radial head.
As we mentioned in most cases, when a patient with untreated enthesopathy comes for the first time, we find features of swelling (decreased echogenicity, blurred echostructure, sometimes thickening) with no signs of increased vascularity. We observe increased vascularization during treatment monitoring, after stimulation of repair processes, after shock wave therapy (about 5-6 weeks after the first treatment, which is usually performed once a week), or administration of platelet-rich plasma (PRP), starting about a week after injection, related to the mechanisms of action of the above-mentioned methods of treatment. Figure 12 demonstrates the technique of PRP injection.

On the surface of platelets, there are growth factors that cause local inflammation, but also promote the formation of new capillaries. Main growth factors include vascular endothelial growth factor (VEGF), transforming growth factor (TGF), connective tissue growth factor (CTGF), platelet-derived growth factor (PDGF),
epidermal growth factor (EGF), and platelet-derived angiogenesis factor (PDAF). Additionally, tissue healing by stimulating the proliferation of fibroblasts and consequently the formation of new collagen fibers is observed. It can be assumed that the injured attachment is healed by the locally induced inflammation [14]. In figure 13 we present the longitudinal evaluation of a 46 year old man manual worker.

**Conclusion**

US is a valuable modality to assess the structures of the lateral elbow joint and surrounding tissues. The main utility is in the diagnostic imaging of typical lateral epicondylitis and in differential diagnosis. US also provides relatively low-cost diagnostic imaging following elbow trauma.

**Conflict of interest:** none

**References**